

USSN 09/690,647

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5. (Amended) The method of claim 4, wherein the inhibitor decreases expression of a gene encoding ERK1/2, a MEK and/or a JNK.
6. (Amended) The method of claim 5, wherein the inhibitor interacts with an ERK1/2, a MEK and/or JNK gene.
8. (Amended) The method of claim 3, wherein the inhibitor decreases the activity of ERK1/2, a MEK and/or a JNK.
9. (Amended) The method of claim 6, wherein the inhibitor interacts with ERK1/2, a MEK and/or a JNK protein.
10. (Amended) The method of claim 6, wherein the inhibitor inhibits ERK1/2, a MEK and/or JNK phosphorylation.

Please insert new claims 19-28:

19. (New) The method of claim 1, wherein the inhibitor is a dominant negative mutant of ERK1/2, a MEK and/or a JNK.
20. (New) The method of claim 1, wherein the subject is overweight or obese.
21. (New) The method of claim 1, wherein the disease or condition is caused, or contributed to, by TNF- α induced lipolysis.
22. (New) The method of claim 1, wherein the disease or condition is caused, or contributed to, by basal lipolysis.
23. (New) The method of claim 22, wherein the inhibitor does not interact with a PPAR- γ receptor and the inhibitor is not sodium salicylate.
24. (New) The method of claim 23, wherein the inhibitor is selected from the group consisting of an antisense molecule, a triplex molecule, a ribozyme and a dominant negative mutant targeted to ERK1/2 or a MEK.
25. (New) The method of claim 1, further comprising determining the level of activity of ERK1/2 or a MEK in the subject.
26. (New) The method of claim 25, wherein the level of activity of ERK1/2 or a MEK is determined in a sample of fat cells from the subject.

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27. (New) The method of claim 1, wherein the inhibitor is administered in the presence of a carrier that facilitates entry of the inhibitor into cells of the subject.
28. (New) The method of claim 1, wherein the inhibitor is administered locally.